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A STUDY OF THE MYOCARDIAL CHANGES IN TWO HUNDRED AND EIGHTY-SEVEN CASES OF ENDOCARDITIS.

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It has long been recognized that prognosis in endocarditis, either of the acute or chronic variety rests primarily not on the valve diseased nor even to a very marked grade on the degree of the valvular lesion, but chiefly on the condition of the heart muscle.

Diagnosis depends mostly on the valve involved and on the nature of the endocarditis, but prognosis on the degree and character of the associated muscle change. One may also say that exact diagnosis in endocarditis is usually relatively easy, but accurate prognosis is difficult and often impossible, since it is as yet impractical to determine or to estimate the real condition of the heart muscle by our present unsatisfactory methods. The valve lesion in aortic disease for example determines the characteristic and easily recognizable physical signs of auscultation, percussion and pulse; but the tension, rhythm, and the maintenance of the circulation rest on the muscular capability and its reconstructive or latent power, factors difficult or impossible of determination.

Treatment is also mostly based on recognition of the dominance of the muscle factor and in nearly all, except possibly syphilitic cases, it is without effect on the valvular change proper, but is directed to the heart muscle and then yields rich reward in the way of successful management of these cardiac cases. Conservation of the muscle and the correct appreciation and control of its more or less limited possibilities in endocarditis is the only line along which successful treatment is to be expected. In spite of this well attested and entirely obvious fact, one cannot but be astonished, in going over the various text-books dealing with diseases of the heart, to find how little attention is paid to the discussion of the changes present in the heart muscle in endocarditis and the manner in which they originate, notwithstanding that in all, treatment is primarily based on the tacit acceptance of the fact that the muscle condition is the most important single factor.

Among the earlier of those who fully recognized this fact, and attempted to systematically study the nature of the muscle changes in endocarditis was Romberg, whose article appeared in the *Deutsches Archiv für klinische Medizin*, for 1893. In this article, Romberg quotes largely from Koester, Stokes, Bamberger, and Oppolzer. Von Leyden, in 1896, also presented an extensive study of the myocardial changes in endocarditis with special reference to those occurring in articular rheumatism. Previous to this, Ribbert[1] conducted experiments designed to show that certain of the myocardial changes developed as the result of embolic processes occurring in the arterioles of the myocardium. Zeigler also shortly previous to this report had advanced the same idea and it remains so stated in his text-book.

On account of the great importance of this subject and because of my personal interest in it, I have made a detailed study of the types of myocardial disease which I have found associated with endocarditis in my autopsy series. The study is based on 236 cases of chronic endocarditis, 35 of acute endocarditis, and 16 of associated acute and chronic endocarditis. These cases were taken consecutively as they appeared in my post-mortem service and unfortunately have not all been studied microscopically, although all instances of questionable or of presumably unusual type have been thus observed. I have further attempted by a preliminary morphological study of the nature of these changes to thoroughly familiarize myself with the condition so that the gross diagnosis has probably in most cases been fairly correct and accurate. As the study has been upon post-mortem findings, no attempt at distinction between obstructive and incompetent valve lesions has been attempted, for the reason that I have found from my associated clinical and postmortem observations that physiological incompetence is often or usually not to be demonstrated at autopsy, being generally due, as Romberg states, to muscular rather than valve defects.

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[1] Ueber experimentelle Myokarditis und Endokarditis, Forts. d. Med., 1886, vol. iv.

At the very outset it became apparent that, as Adami and Nicholls[2] state, the heart muscle changes are not usually primarily nor even secondarily dependent upon the endocardial disease. This is especially true of those instances in which a dominant toxic factor was present. In numerous cases the myocardial changes have been concomitantly caused by the very same elements which were concerned in the production of the endocarditis.

Thus, 13 instances of associated endocardial and myocardial disease occurred in general simple infections, 14, in syphilis, 16 in tuberculosis, 1 in mixed infection following typhoid fever, and 1 in epidemic cerebrospinal meningitis. By far the larger number, however, appear to have originated in those general conditions in which a reduction of general body resistance probably first produced the myocardial disease and, perhaps, favored secondarily endocardial changes, as for example, in 61 cases of nephritis, 9 of neoplasm, 4 of grave anemia, 3 of diabetes, and in 56 instances of alcoholism. In regard to the connections between alcoholism and myocardial disease associated with endocarditis, I wish to state as I have done before, that I fully agree with Cabot that alcohol does not *per se* induce cardiac disease, but I am convinced that the associated conditions which go to make up the composite picture of alcoholism do.

The ways in which the heart muscle may become involved in endocarditis may, perhaps, be satisfactorily summarized under the following chief heads.

1. It may become diseased as a common result, of the process or condition under which the endocarditis itself develops. For example, the acute parenchymatous degeneration of the heart muscle, which is the most frequent myocardial change developing in cases of rheumatic fever, doubtless arises from the same toxic cause, be it bacterial or otherwise, which determines the inflammatory process of the valve surfaces. Undoubtedly under this heading are comprised the most frequent lesions of the heart muscle associated with endocarditis.

2. It may result from the embolic plugging of one of the coronary vessels from the dislodgment of necrotic material from the diseased valve, from embolism by lodgment of the bacteria circulating within the blood stream which may have caused the endocarditis or from hyaline thrombi. As a result an ischemia or infarction of the heart muscle follows, succeeded by necrosis and usually after a few days by cardiac rupture, or if an active fibrosis occurs in the diseased area a cicatricial scar or aneurism may follow. This method of origin of muscle disease has been abundantly confirmed by the researches of Zeigler, Romberg and Ribbert, and was found present in 5 cases of my series.

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[2] Principles of Pathology, vol. ii.

3. The muscle may become involved as a result of ischemia following obstruction at the opening of the coronary vessels or in the course of their lumen. Obviously this form of heart muscle change is most frequent in aortic disease and as stated by Osler may also follow from a deficient aortic pressure quite independent of coronary disease. No instances of this last character were observed, nor were any seen of obstruction of the coronary vessels without arteriosclerosis. Eight instances definitely due to coronary arteriosclerosis were found, however, nearly all occurring in aortic disease.

4. It may become diseased by the direct extension of the ulceration from the point of valve disease into the myocardium. Ulcerative and fibrotic invasion of the bundle of His in this manner has been of late frequently reported and the condition has been especially studied by Monckeberger.[3] No recognized cases of this character are included in this series, although at present, I have two probable cases of this nature under observation.

5. Myocardial degeneration or inflammation either acute or chronic may be directly set up as a result of a superjacent endocarditis. This develops most certainly in cases of mural endocarditis, although it may also occur in simple valvular lesions and invasion of the papillary muscles is especially likely to take place in such instances. Involvement of the papillary muscles is extremely frequent especially in the chronic cases, and is often largely responsible for valve incompetence. Definite mural endocarditis with myocardial disease was found in but one case of my list.

6. Involvement and fibrosis of the muscle may readily originate in the papillary muscles whose integrity has been first compromised by the overstretching which follows cardiac incompetence or relaxation of the valve rings. This may be followed by a more or less generalized fibrosis extending out from these foci, a condition which I have frequently observed.

7. Disease and especially fibrosis of the auricles undoubtedly follows the auricular distention dependent on ventricular irregularities due to disturbed muscular conductivity and arrhythmia.

8. Banti, according to Adami, has shown that venous stasis, such as occurs very commonly in various diseases of the heart including valve lesions, may, in itself produce changes in the muscle bands which become of grave import. I have never definitely recognized this process.

9. Probably most frequent of all is disease of the myocardium, notably that of the ventricles, which occurs from other causes entirely independent of the endocarditis. This was definitely the case in 81 of the 176 instances of associated endocarditis and myocardial disease in my 187 cases of endocarditis.

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[3] Untersuchungen über das Atrio-ventricular bundle im Menschlichen Hertzen, Jena, Fischer, 1908.

10. There can be no doubt but that other cases occur in which the myocardial lesions are definitely dependent on the primary endocardial disease, these instances, are, however, from their varied nature, difficult or impossible of definite post-mortem recognition.

As to the character of the valvular lesions found in the group of 287 cases, I was much surprised to find that an association of aortic and mitral endocarditis was by all means the most frequent. This combination was found in 152 cases, nearly three times as frequent as pure aortic disease and over six times as common as pure mitral lesions. This is not in accord with statistics published by most other observers. Osler gives in a series of 209 cases, aortic and mitral, 41; aortic alone, 53; mitral alone, 77; tricuspid, 19; pulmonary, 15; heart walls, 33. My list further gives: 40 instances of chronic aortic lesions, 10 of acute and 2 of acute on chronic; 12 of chronic mitral lesions, 6 of acute and 4 of acute on chronic; 6 associated lesions of the aortic, mitral and tricuspid; 5 of aortic, mitral and pulmonary lesions, all except one of the chronic variety. In only 1 chronic instance were all the valves involved. The aortic and tricuspid were involved together in 3 chronic cases. The pulmonary and aortic valves were involved in 3 chronic, but no acute cases, and the mitral and tricuspid in a single instance each, in the chronic, acute and acute on chronic lists. The pulmonary valve alone was diseased in 3 chronic cases. The tricuspid alone was involved in 1 chronic and 2 acute cases. In but 1 acute instance was a mural endocarditis without a valve lesion present, although in many instances mural changes of an inflammatory and degenerative character were demonstrable in all forms of valvular endocarditis.

From all the various ways by which the muscle becomes diseased, the number of distinct anatomical changes which result are relatively few. By far the most abundant numerically as well as important physiologically, are changes of a degenerative type, which, although they are quite frequently classified as inflammatory and included under the heading of myocarditis, are really degenerative in their nature. In the entire series of 278 cases, true inflammatory change in the heart muscle was found in but 6 instances, exclusive of those cases showing fibrosis which obviously may be either degenerative or inflammatory in origin. On the other hand, muscle changes of a definitely degenerative character were found in a total of 150 instances, exclusive of those showing purely fibrotic alterations.

As was to be expected and in full accord with well established clinical observation, concerning the relative innocence of mitral endocarditis, the rate of occurrence of disease of the muscle in mitral lesions stood last of all. Among the chronic cases of mitral disease in but 5 out of the total of 12 was muscle disease of serious nature demonstrable. In 4 of these cases only was the change sufficient to permit the cardiac lesions being justly classified as among the chief causes of death. This point indicates an apparent interdependence between the degree of muscle disease, or the absence of it, in determining prognosis.

In sharp comparison to mitral disease are the 52 instances of aortic lesions, 18 of which died as a direct result of the cardiac disease and in all except 15, of both cardiac fatal and non-fatal cases, definite muscular changes were present. In all the 10 instances of acute aortic lesions the heart muscle was grossly diseased, and the cardiac lesion was rated as among the chief causes of dissolution. This indicates what has long been recognized, namely, that aortic lesions are usually the most serious of those of the endocardium. The high percentage of muscular involvement in aortic endocarditis is probably partly due to the greater mechanical muscle strain present in this disease.

In cases of associated chronic aortic and mitral disease but 22 of the 128 died as a result of the cardiac disease; yet in these same instances, the muscle was definitely and grossly diseased in all but 35 instances. Of the 15 acute associations of aortic and mitral lesions, the heart muscle was gravely involved in all except 5 instances.

As to the relationship between the valve lesion and the type of muscular change, I find that in associated aortic and mitral lesions 36 cases show purely fatty changes, 9 fatty and parenchymatous and 15 purely parenchymatous; 17 showed interstitial changes. In but a single instance was a simple muscular hypertrophy without degenerative alteration present. In the group of pure mitral disease, the number of cases was too small, only 5 out of 12, to permit conclusions of any definite value to be drawn, but here again, fatty degeneration led with 3 out of the 5 cases.

It was confidently expected that at least some examples of pure simple hypertrophy would be found in the group of chronic aortic disease, but on the contrary, as before stated, the highest percentage of muscle disease was found in this list. Death from cardiac lesions was also more frequent in these than in any other series of cases. Six instances of fatty degeneration were found, 6 of pure parenchymatous, 2 of fatty degeneration and brown atrophy, and 5 of brown atrophy alone. But a single instance of purely fibrotic change was present, but interstitial alterations were associated with fatty degeneration in 2 instances and with brown atrophy in 1. In the acute cases of aortic disease simple hypertrophy was present once, parenchymatous degeneration, twice; parenchymatous and fatty, once; fatty, once; brown atrophy and fatty, once; interstitial, twice; and inflammatory or true myocarditis once.

In the entire group of chronic cases pure parenchymatous changes occurred but twenty-five times, while fatty changes were present in 67 instances, associated fatty and parenchymatous in 17. I was somewhat surprised at finding in the entire series of chronic cases only 22 instances of pure interstitial change, 17 of which, as before stated, occurred among the associated mitral and aortic lesions.

From this series, it appears that the most frequent myocardial lesions occurring with endocarditis are degenerative in character. Of these, fatty degeneration is the most frequent. In this general study no attempt has been made to present unusual and rare conditions, such as hyaline and amyloid degeneration but the object has been to obtain a general idea only of the most frequent changes to be expected in the heart muscle in the course of endocarditis.

It is, therefore, unnecessary in this place to go into a discussion of the various types of myocardial degeneration or to elaborately describe the changes present in them. For our purposes, it is, perhaps, sufficient to point out that fatty degeneration occurs in most cases as a sequence of an unhealed parenchymatous degeneration and parenchymatous degeneration of greater or lesser grade is present in the heart muscle in practically every instance of toxic disease, unhealed cases of which then pass over into the fatty change. Munk[4] has shown that myaline and protagen are formed in fatty degeneration of the heart muscle just as in other types of this degeneration elsewhere, so that in all probability the change is precisely like that occurring in voluntary muscle. This work has been verified by A. Erlandsen. Monckeberg found fatty degeneration of the atrioventricular bundle and according to him this type of degeneration is apt to be succeeded by calcification and fibrosis. It is, therefore, highly probable that fibrosis occurs usually as a sequence of a preliminary fatty degeneration which would account for the frequent association of these changes.

CONCLUSIONS. Myocardial disease is present in greater or less degree in practically all cases of endocarditis acute or chronic.

The type and degree of these changes determine to a large degree, the possibilities and the future of any case of endocarditis.

The most frequent changes in the myocardium in association with endocarditis are degenerative ones. Inflammatory lesions are relatively rare.

In most instances the myocardial change is not determined by, nor is it the direct result of the endocarditis, but is caused independently by the same, affiliated, or by entirely independent conditions.

The most frequent form seen in chronic or late acute cases is fatty degeneration, a lesion which differs in no material histo-chemical respect from that involving voluntary muscle. From analogy, it seems that this degeneration in most instances succeeds a primary parenchymatous alteration. As a sequence to fatty degeneration, fibrous replacement and probably brown atrophy frequently appear.

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[4] Diss., Berlin, 1907.

There is no regular association between the special valve lesion and the type of associated myocardial degeneration, except that these changes appear more constantly and in most serious grade in aortic than with other lesions, probably because of mechanical reasons.

REPORT OF A CASE DEMONSTRATING PULSUS ALTERNANS, BLOCKED AURICULAR EXTRASYSTOLES, AND ABERRANT VENTRICULAR ELECTRIC COMPLEXES.

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The following case is of interest because of the presence of alternation in the pulse, the occurrence of blocked auricular extrasystoles and an aberrant type of ventricular complex in the electric curves.

History. J. H., aged forty-five years, a widower, is employed as a butler. He was admitted to University College Hospital, London, on April 1, 1911. His father is aged seventy-six years; hale and hearty. His mother died of nephritis, aged sixty years. One brother died of rheumatic fever, aged thirty-five years, another of diabetes, aged twenty-eight years, and a third, of cirrhosis of the liver, aged forty-two years. A sister died in early life of a disease unknown to the patient. Three brothers and a sister are still living and well. As a child the patient had measles; later, at the age of eight years, diphtheria. Scarlet fever, chorea, rheumatic fever and tonsillitis are denied.

When aged twenty-five years, the patient suffered from an atypical attack of pneumonia. Its duration was ten days; the fever declined by lysis and convalescence was prolonged. Ten years later the patient had an attack of severe pain in both calves. There was no fever and no joint involvement. This attack lasted eight days and he remained in bed three days. He has had repeated attacks of "influenza." The first occurred at the age of twenty-four years, the second when aged forty-one years, and the third and last attack occurred twenty months ago. Each attack lasted approximately ten days. No complications are known to have followed the first two of these attacks.

All of the symptoms relative to the patient's present condition date from the last attack. Immediately after it he experienced dyspnea on exertion, mild but persistent cough and general weakness. Attacks of palpitation were apt to occur after exertion, excitement, or gastronomic indulgences. His general health remained fair, however, and he was able to pursue his work until some months ago, when his symptoms became exaggerated, persistent dyspnea being the most distressing symptom. Pain was never present.

Up to two months ago the patient was a moderate drinker and smoker—one or two glasses of ale per week and three pipes daily. Since the onset of this attack he has neither drunk nor smoked. Patient denies all venereal infection. No history of syphilitic manifestations is obtainable. Bowels regular; appetite good; sleep fair.

State on Admission. The patient is a fairly well-nourished man. The mucous membranes are cyanotic. There is considerable dyspnea. The thorax is markedly rachitic.

Respiratory System. The lung-liver border in front is at the sixth rib. The lower limit of the lungs posteriorly is at the tenth thoracic vertebra. The borders alter little with respiration. The percussion note is clear over the whole chest. On auscultation, numerous sibilant and sonorous rales are heard. Over a circumscribed area 4 cm. in diameter, and situated in the third and fourth intercostal space in the midclavicular line of the right chest, numerous fine crepitations are audible. Expiration is universally prolonged.

Cardiovascular System. All palpable vessels are soft. The pulse is markedly irregular; its rate per minute is 85 to 100. The systolic blood pressure is 150 mm. Hg. The apex beat is felt in the fifth intercostal space in the midclavicular line, and is diffuse and wavy. There is no thrill. The right limit of cardiac dulness lies 4 cm., the left limit 11 cm. from the mid-sternal line. Upper limit of cardiac dulness is at the third rib. There is marked epigastric pulsation. The sounds at the apex are dull and distant. A soft systolic murmur is present. The second aortic sound is accentuated.

Abdomen. Liver, spleen, and kidneys are not palpable. No ascites present.

Extremities. There is slight edema of the lower extremities.

Nervous System. Negative.

Urine. Acid; traces of albumin; no sugar. In the sediment there are occasional granular casts, a few white blood corpuscles and amorphous urates.

Sputum. The sputum was negative for tubercle bacilli on three successive occasions.

During his stay in the hospital the patient's pulse became more regular. The systolic murmur disappeared. The edema cleared up, and the breath sounds became free of rales and crepitations. The blood pressure fell to 100 to 110 mm. Hg and stayed there. All subjective symptoms were markedly relieved.

The Polygraphic Tracings. The pulsus alternans is well shown in the accompanying tracings (Figs. 1 and 2). As is usual, when pulse alternation is combined with extrasystoles, the alternation is more marked after the premature beat. It may be noted that, as